lation.   The peculiar character of these malformations is pointed out by Dr. Montgomery, and ample directions given to prevent a mistake in diagnosis. The chapter forms a very appropriate termination to a book unusually rich in practical suggestions.   J. C. D.

Art. XIX.—*Traité Expérimental et Clinique d'Auscultation appliquée à l'étude des Maladies du Poumon et du Cœur.*   Par le Docteur J. H. S. Beau, Médecin de l'Hôpital Cochin, Agrégé Libre de la Faculté de Médecine de Paris, etc. etc.   A Paris: Chez Baillière, 1856.   8vo. pp. 628.
Treatise, Experimental and Clinical, on Auscultation, as applied to the study of Diseases of the Lungs and Heart.   By J. H. S. Beau, etc.

The author of this work is not a new candidate for distinction in the annals of medicine.   Any one tolerably conversant with the current medical literature of France, for the last twenty years, must be familiar with the name of M. Beau, as connected with a variety of questions pertaining to clinical researches.   Few observers, in fact, are more frequently referred to by the French medical writers during the period just mentioned; but, if the recollections of the reader enable him to recall the scientific questions with which the name is associated, he cannot fail to be struck with the fact that, in general, writers have been accustomed to refer to the views of M. Beau, in order to dissent from or endeavour to refute them.   Such, at all events, is the impression derived from our own reading of French medical works, and we have long since been led to expect, almost as a matter of course, whenever we meet with a citation of any of his opinions, to find that they are not in accordance with those of the author.

From the work which we now propose to review, we learn that the previous contributions of M. Beau to medical literature, have consisted in communications to the medical journals of Paris, and that they are, for the most part, contained in the *Archives de Médecine.*   His articles have been numerous, falling but little short of fifty.   Of this number, fifteen relate to diseases of the lungs and heart, being mainly devoted to questions pertaining, directly or indirectly, to auscultation as applied to the diagnosis of these diseases.   The object of the present work is to reproduce and bring together the views contained in these fifteen memoirs, which have appeared, from time to time, during the last twenty years.

The work is not designed to be a complete exposition of the principles and practice of auscultation, but mainly to present and advocate anew certain doctrines peculiar to the author, and opposed to those which are commonly received.   This being professedly the scope and character of the work, we shall regard it, as a reviewer, in that light.   We purpose to direct our attention chiefly to the more prominent of these peculiar doctrines, and to analyze, with a due regard to brevity, the claims which the author sets forth in their behalf.

The author solicits of the readers of his work that they will, if possible, read it unbiassed by preconceived notions, and with the sole aim to discover the truth.   He has a just appreciation, in the abstract, of the difficulty with which truth makes progress when opposed by settled convictions.   What discoverer of new truth has not had abundant cause for impatience and dissatis-

faction at that principle of conservatism in the human mind which holds on with dogged tenacity to well-established error, fearful of novelty and innovation! There is, however, another phase of mental conservatism which may have presented itself less forcibly to the reflections of M. Beau—it is the pertinacity with which the mind adheres to its own creations. How seldom is it that the originator of new views is ever led to abandon them! How rare are the examples of authors who, publishing at different epochs, ever acknowledge the errors contained in their previous works; on the contrary, is it not an almost stereotyped mode of expression for an author to say that further observation, or experience, or reflection have only served to confirm the correctness of the conclusions to which he has been heretofore committed? If an assent to error involves an obstacle to the reception of new truth which is difficult to be overcome, how much greater is the prejudice in behalf of error when the latter has been not adopted but created by the mind which believes it to be true—the obstacle is then almost insuperable! We shall see whether the work before us furnishes occasions to illustrate most the tardiness with which scientific discoveries obtain credence, or the blind partiality which leads an author to continue to maintain, with zealous enthusiasm, peculiar doctrines that have been abundantly disproved.

M. Beau divides his treatise into two parts: the first part treats of auscultation of the lungs, and the second of auscultation of the heart and vessels. One-third of the volume is devoted to the former, and the remaining two-thirds to the latter. Each part is subdivided into two sections: the first section treating of physical signs, and the second of the different diseases, respectively, in their relations to these signs. We proceed to notice the portion of the work devoted to auscultation of the lungs.

The vocal sounds, normal and abnormal, occupy but little space. They are disposed of in about four pages. As regards the mechanism of the vocal sounds heard over the chest in health and disease, the author recognizes the principles of reverberation and conduction only, making no reference to the theory of consonance advocated by Skoda. Under the head of the normal sounds, the only novel statement which we observe is, that when the voice is produced by an act of inspiration, which it is asserted can be done after some practice, the pulmonary resonance is much more marked than when words are pronounced by an expiratory act. This fact we have not verified. Practically, however, it cannot be made of much service, inasmuch as in the cases of disease which present themselves for examination, we shall not find that patients are prepared by the necessary practice to be able satisfactorily to reverse the habitual mode of speaking during expiration.

Bronchophony and pectoriloquy are regarded merely as different degrees of resonance. This is the view taken by other authors; yet, certainly the distinction in character between these two signs is sufficiently defined. In bronchophony the *voice*, in pectoriloquy the *speech*, is transmitted. Now, not only may the voice be abnormally loud and near the ear of the auscultator without any transmission of speech, or, in other words, an appreciation of articulated words, but the pectoriloquy may be complete without intensity of vocal reverberation. In short, pectoriloquy may exist without bronchophony, as well as the latter without the former. This distinction is not generally made by practitioners who employ, more or less, physical exploration, a fact not to be wondered at, since the same remark is applicable, for the most part, to authors treating specially of physical diagnosis.[1] It must, however, be

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[1] A distinction, hardly less marked, exists between merely increased vocal resonance and bronchophony. In the former, the reverberation or resounding of the

admitted that the distinction, in a practical point of view, is much less important than it would be, were it true, as was supposed by Laennec, that pectoriloquy is a diagnostic sign of a pulmonary cavity.   M. Beau adds his testimony to that of others, to the occurrence of perfect transmission of speech in certain cases of pulmonary solidification.   No mention is made of the transmission of whispered words, which, according to Walshe, is highly significant of an excavation.   Our own observations, however, have led us to a different conclusion.   In fact, we have oftener met with instances of solidification in which words spoken in whispers were transmitted, than with those in which ordinary pectoriloquy existed.

Without discussing the mechanism of ægophony, the author states that it does not pertain exclusively to pleurisy, but occurs occasionally in pneumonia. The united testimony of different observers has sufficiently established the correctness of this statement.   He states that the sign may be produced by compression of the bronchi or trachea.   He has observed it twice in cases in which this compression arose from aneurism of the aorta.

The respiratory sounds are considered at much greater length.   Here we come to theoretical views which are peculiar, and on which he bestows considerable discussion.   He embraces the normal respiratory sounds under the head of *souffle glottique.*   In 1834, he submitted the theory that the tracheal, bronchial, cavernous, and vesicular respiratory sounds are all due to those produced in the upper respiratory passages; viz., the nostrils, pharynx, and larynx.[1]   Authors of works on physical exploration, in the mean time, have generally referred to this theory and devoted to it consideration sufficient, in their judgment, to disprove it, but not enough to convince M. Beau of its incorrectness.   So far from this, in 1840, he argued its merits in a paper contained in the *Archives de Médecine;* and, in the present treatise, he discusses the point still more elaborately, and with undiminished zeal.

This theory has found but few advocates.   Dr. Spittal, of Edinburgh, has been its ablest supporter.   Dr. Spittal, however, regards the sounds heard over the chest as laryngeal exclusively, and not guttural, as contended by M. Beau in his first publications.   The latter, in the present treatise, appears to adopt the views of Dr. Spittal in this respect.[2]   Dr. Spittal, availing himself of the three bronchial divisions of the trachea in the sheep, contrived an apparatus, by means of which he professed to demonstrate that the sounds emanating from the pulmonary vesicles are, in reality, produced in the larynx. To give an account of this apparatus would require too much space, and too tedious details.   It is described and figured in the work before us.   Such demonstrations are open to two important objections: 1. Artificial contrivances out of the body cannot be taken as representing the pulmonary organs *in sitû* and in vital action; or, at all events, inferences drawn from the former and

voice is greater than in health; in the latter the voice is abnormally transmitted, that is, nearer the ear of the auscultator.   Here, too, as in the case of bronchophony and pectoriloquy, the vocal resonance may be greatly increased without bronchophony, and *vice versa.*

[1] M. Beau acknowledges his indebtedness for this theory to M. Chomel, who propounded an inquiry in 1824 whether the respiratory sounds might not admit of such an explanation.   M. Chomel, however, makes no reference to it in the last edition of his work on Pathology, issued during the present year.

[2] The author, however, is careful to claim that he has not deviated from his original views.   He says the title *bruit guttural* was given rather to indicate the approximative than the actual seat of the sound.   We should have not thought less of his impartiality had he evinced less tenacity in maintaining his consistency.   However it may be in politics or morals, in science consistency is not always a jewel.

applied to the latter, are to be received with great caution. It is impossible for art to compass all the elements involved in the performance of respiration. We can never be certain either that nothing essential has been omitted, or that something giving rise to important modifications has not been superadded. In view of this objection, such experiments are only entitled to a certain degree of weight as corroborative proof in behalf of conclusions based on observations made on the living body. 2. Contrivances which are somewhat complicated, like the apparatus of Dr. Spittal, require too much trouble for the experiments to be repeated by many; and, hence, we have limited testimony as to the results. Now it is, alas! but too true, that with no other disposition than that of truth-seeking, a mind biassed in favour of certain views, is liable to judge, even in matters of plain observation, by impressions received through a distorted medium. Honesty of purpose, although a *sine qua non*, is not the only requisite for a faithful observer. Freedom from preconvictions is hardly less essential. Here lies the secret of a large proportion of the false facts with which medical science abounds!

Exclusive of Dr. Spittal's experiments, on what does M. Beau base his theory? So far as the results of direct observation are concerned, mainly on the fact that when the sounds produced in the upper passages, *i. e.*, chiefly at the glottis, are voluntarily suspended in respiration, the respiratory murmur ceases to be heard on auscultation, and *per contra*, the latter are developed in proportion as the former are intense. Now, in the first place, it is difficult, if not, indeed, impossible to determine by direct observation, this relation between the sounds produced at the glottis and those heard over the chest. Auscultation cannot be practised in both situations simultaneously, and every auscultator must be aware that the respiratory sounds are by no means uniform as regards intensity with successive acts of respiration. They are constantly fluctuating in this respect. It is, therefore, evidently a theoretical assumption to say that when the vesicular murmur is feeble or absent, it is in consequence of a corresponding diminution or deficiency of a sound generated at the glottis. That, in general, the intensity of sound in these situations should correspond, is a rational inference from the fact that the force of the current of air, whether weak or strong, will affect equally the superior and inferior air-passages. It is difficult, indeed, to understand how the sounds at the glottis can be voluntarily repressed or suspended without, at the same time, diminishing the force of the current of air in the bronchial tubes and vesicles. The possibility of doing so, although assumed without discussion by M. Beau, certainly is questionable. But there is a mode in which observation may be made to bear upon this point; viz., by comparing, in different individuals, the relative intensity of the respiratory sounds in the larynx and over the chest. Is it found to be a rule that in persons with loud laryngeal respiration the vesicular murmur is also intense, and *vice versa?* This should be the case if the theory of M. Beau be correct. We happen to possess a collection of observations made on healthy persons in whom the sounds heard over the larynx, trachea, bronchi, and pulmonary organs were recorded, without any reference to this question. On referring to these notes, a rule of correspondence as regards the intensity of the sounds in the upper and lower passages, in habitual respiration is found not to hold good. If such a rule exists, the exceptions are so numerous as to disprove the dependence of the latter on the former.

The characters which belong to the vesicular murmur, contrasted with those of the laryngo-tracheal respiration, it would seem, should alone suffice to disprove M. Beau's theory. The laryngo-tracheal respiration embraces a sound

during inspiration and during expiration; the latter being more intense and prolonged than the former, if the respiratory acts are forced. The vesicular murmur very often consists solely of an inspiratory sound, and if a sound of expiration be present, it is short and feeble. M. Beau thus accounts for this difference :—

"The *bruit glottique* during inspiration penetrates the vesicles with the current of air which goes to fill them, and divides itself, so to speak, into an infinite number of minute isolated *bruits* which seem to be produced beneath the part of the thorax where they are perceived by the ear; on the other hand, the *bruit glottique* during expiration tends to reverberate through the pulmonary parenchyma at the moment when the air which conducts the sound leaves the vesicles. The *bruit* therefore gives rise to are verberation which is very imperfect. It gives the impression of a confused echo occurring in the large tubes, which with difficulty traverses the left parenchymatous layer of lung which intervenes." (P. 18.)

Notwithstanding this explanation, if the vesicular murmur were, in fact, produced at the glottis, and transmitted by reverberation, we should expect the expiratory sound to be oftener present and more intense than it is found to be in view of the greater intensity and direction of this sound over the larynx.

That the receding current of air during expiration is not an insuperable obstacle to the transmission of sound from the superior air-passages, is shown by the distinctness with which the whispering *souffle* is heard over certain portions of the chest in health. But, assuming that the *bruit glottique*, in other words, the laryngeal respiration, is transformed into the vesicular murmur by reverberation, how is the fact to be explained that the inspiratory sound is longer in the latter than the former? Does the sound become prolonged by being transmitted? How is the variation in pitch to be accounted for? The vesicular murmur is notably lower in pitch than the laryngeal respiration. Again, how is it that the *bruit*, during its transmission, undergoes such a marked change in timbre or quality? In the latter respect, the vesicular murmur differs in a striking manner from the sound heard over the larynx. We shall content ourselves with merely suggesting these points, without arguing them. Indeed, it seems almost superfluous to discuss the merits of this theory since experimental observation on the living and dead body appears to have demonstrated its incorrectness. In cases in which tracheotomy is practised, the *bruit glottique* is, of course, suspended, yet the vesicular murmur is found to continue. MM. Barth and Roger give a report of experiments in which the trachea was divided in inferior animals; respiration, carried on without the air passing through the larynx, nevertheless was accompanied by the vesicular murmur. Artificial respiration, after death, through the trachea separated from the larynx, was accompanied with the same result. M. Beau attempts to dispose of these experiments by supposing that an abnormal sound was produced at the aperture into the trachea, or the divided extremity of the latter, and that this sound replacing the *bruit glottique* gave rise to the respiratory murmur by transmission. But such a sound did not exist in all the cases, and when it was observed it differed from the sound produced at the glottis while the vesicular murmur was unchanged. Moreover, MM. Barth and Roger state that, in one instance, this sound was in fact transmitted, and could readily be distinguished from the coexisting vesicular murmur.

Our readers will perhaps think that we have devoted too much space to the notice of this theory. We should be disposed to enter into a full discussion of

it, if by so doing we could hope to relieve authors of works on auscultation of the apparent necessity to devote more or less space to a refutation of its claims to be regarded as the exposition of the mechanism of the normal respiratory sounds. We will add a single consideration, which occurs to us at the moment. If the vesicular murmur be due to the transmission of the *bruit glottique*, the intensity of the former should gradually diminish in proceeding downward over the chest. The author, in fact, states this to be the case, and adduces it as an argument in favour of his theory. Now, in certain persons the vesicular murmur at the base of the chest behind is quite as intense, if not more so, than at the summit of the chest in front. Auscultation of the chest in a series of healthy subjects will supply the proof of the correctness of this assertion.

The inquiry, "What is the mechanism of the vesicular murmur?" were we to pursue it, would lead to an extended discussion. We cannot, however, forbear bestowing on it a few words. That the murmur is produced solely by the friction of the air, in its passage into and from the vesicles, as supposed by Laennec, we do not believe. The truth probably is, that the air in the vesicles and smaller tubes is changed but to a limited extent as a direct consequence of the respiratory movements. On this point, Lehmann says:—

"We should form a very erroneous idea of the motion induced by this mechanism (the respiratory movements), were we to conceive that it was able to agitate the whole of the air contained within the cavity of the chest. For even when the contraction is relatively considerable, only a small fraction of the air is expelled, and an equally small proportion admitted by its expansion ; hence it is only in the wider air-canals that the air can be absolutely changed, whilst in the narrower vessels there is only an undulating current of the stagnant air-column, induced by the contractility of the walls. The change, therefore, depends solely upon the different degrees of diffusibility of the gases. However simple this latter circumstance may appear, Vierordt has the merit of being the first who experimentally illustrated these physical relations."[1]

It is surprising that M. Beau has not availed himself of this view, in order to support his theory, indirectly, by disproving that commonly received. We would submit, as the most rational explanation of the vesicular murmur, that it is produced by the separation of the walls of the cells and intercellular passages which, to a greater or less extent, come into contact as the lungs collapse in expiration. The lungs collapse in this respiratory act, it is true, but partially; nevertheless, a certain proportion of the cells must be more or less deprived of air. The disruption of the surfaces in contact and slightly adherent, when the cells are dilated in the act of inspiration, gives rise to the vesicular murmur, or, at all events, is the source of the peculiar character of sound (the vesicular quality) by which this murmur is distinguished. This hypothesis will serve to account for the vesicular murmur being so often heard only during inspiration, and for the absence of the vesicular quality in the expiratory sound when the latter is heard. It will account, also, for the prolonged duration of the inspiratory sound. It enables us to understand why the vesicular murmur is intense in proportion to the duration and force of the preceding expiratory act; as, for instance, directly after coughing, the walls of the cells coming into contact to an extent proportionate to the completeness of the expiration and consequent collapse of lung. Moreover, the greater intensity of the vesicular murmur in children (puerile respiration) is rendered intelligible by this hypothesis. The greater mobility of the thoracic walls permits a greater extent of collapse of lung in the expiratory act in early life.

Regarding the normal respiratory sounds and their abnormal modifications

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[1] Physiological Chemistry, Am. ed., vol. ii. p. 428.

as emanating from the larynx, M. Beau distinguishes them as the superior or supra-clavicular sounds, and he applies to the adventitious pulmonary sounds the names *inferior, infra-clavicular,* or *thoracic.*   The latter he divides into *intra-pulmonary* and *extra-pulmonary.*   The former include all the bronchial and vesicular râles, and the latter the pleural or friction sounds.   We proceed to notice very briefly certain positions which the author takes in this portion of the work.

The division of bronchial râles into *vibrating* and *bubbling* he claims as original.   It is generally attributed to M. Raciborski.

Of vibrating râles he recognizes the varieties generally adopted, viz., sibilant and sonorous or snoring; and he adds a new variety, which he calls a *râle insonore.*   The intrinsic characters of the latter he does not distinctly point out.   It is apparently an approximation to, rather than a well-developed *râle.* The title implies this. He states that it may be distinguished from the normal vesicular murmur by its being more or less circumscribed in the area over which it is heard with its *maximum of intensity.*   We have no difficulty in understanding the sign to which he refers.   The propriety of considering it as a distinct râle may be doubtful; but it should be understood, in order to avoid errors. It is sometimes confounded with an abnormal intensity of the vesicular murmur, and hence the latter is said to belong to certain cases of bronchitis. Again, it is confounded with what is called *rude* respiration, the latter properly denoting an approximation to the bronchial respiration.[1]   Thus confounded, its pathological significance is liable to be misinterpreted. Dr. Bowditch applies to a sound, which we suppose to be the same, the title of "mucous respiration," and points out its occurrence in connection with the circumscribed bronchitis occurring in proximity to a tuberculous deposit.

The vibrating râles, according to M. Beau, are produced solely by an obstruction of the bronchial tubes from the presence of mucus, and are never due to swelling of the mucous membrane.   The reason which he assigns for the latter statement is, that a swelling of the membrane must persist for a certain period, and this cannot be reconciled with the constant variations from one moment to another which characterize these râles.   We can conceive, however, of local congestions occurring within the bronchial tubes, diminishing more or less their caliber, or producing complete occlusion, and disappearing in a short time, even lasting but a few moments.   Every one has experienced transient stuffing of the nostrils from some accidental cause which comes and goes within a very brief period, being sufficient, while it is present, to prevent respiration through these passages; and if this may occur here, there is no good reason why it may not in the bronchial tubes.   We think that M. Beau is in error in denying that the râles are ever produced by swelling of the membrane; but that they are generally caused by the presence of tenacious mucus is probable. A test of their being due to the latter is their disappearance or transference to another situation after an act of coughing.

The bubbling râles he divides into the gurgling, mucous, moist crepitant, and dry crepitant.   The moist crepitant râle is that generally known as a subcrepitant or a fine mucous râle, being produced within the minute bronchial branches. The dry crepitant râle is the true crepitant characteristic of the first stage of pneumonia.   This distinction, based on the sensation of moisture in the one case and dryness in the other, is well founded.   The author is led to doubt whether

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[1] M. Beau applies to the modified respiratory sounds heretofore called generally, by writers on physical exploration, *rude,* the name *tubo-vesiculaire.*   The writer has suggested a name analogous in its significance, viz., *broncho-vesicular.*

the dry crepitant is a bubbling râle, and suggests that it is owing to an abnormal dryness of the air-cells, incident to the early stage of inflammation. He says:—

" I should not hesitate to adopt this manner of accounting for the production of the dry crepitant râle, if it were fully demonstrated that inflammation occasions dryness of the pulmonary vesicles."

M. Beau has probably never heard of the ingenious and beautiful explanation of the crepitant râle submitted by our countryman, Dr. Carr. We had the pleasure of stating it to one of the most distinguished of the Parisian teachers, and we were gratified with the readiness with which he admitted it to be the most satisfactory theory yet offered. We have a right to presume that our American readers are familiar with it, and, therefore, that it would be supererogatory to reproduce it here. M. Beau appears to think, because the sound is characterized by dryness, that it must denote an abnormal dryness of the parts in which it is produced. But this is a *non sequitur*. Nothing can be drier than the sound produced by alternately pressing together and relaxing the pressure of the thumb and finger moistened with a little paste or gummy solution. The dryness, when the crepitant râle is in this way imitated, is not less marked than when a lock of hair is rubbed between the thumb and finger, near the ear, after the plan proposed by Dr. Williams. We do not doubt that Dr. Carr's explanation will, in time, be adopted, and, as a matter of personal reputation, we would rather be entitled to the merit of having originated it, than to have been the author of any one of the theories peculiar to M. Beau.

The crackling sounds, occurring in cases of pulmonary tuberculosis, the author regards as belonging either in the class of humid or dry crepitant râles; and in this opinion we believe him to be correct.

With reference to the three varieties of *crumpling* sounds (*froissement pulmonaire*), as described by Fournet, we are gratified to find that M. Beau offers the same explanations of two of them which we have elsewhere done.[1]

The *bruit de cuir neuf* he regards as neither more nor less than a pleural friction sound, and the *bruit plaintif gémissant* is a vibrating râle. The *bruit rapide et sec que l'on obtient en soufflant sur du papier sec*, or a tissue-paper sound, he supposes to be a transmitted *souffle glottique*. The last-mentioned explanation is open to the objections which apply to the author's theory of the normal respiratory murmur. The sound is probably simply a crepitant râle.

Under the head of the extra-pulmonary, or pleural sounds, we find nothing peculiar to the author, and we proceed to pass rapidly in review the second section, devoted to pulmonary diseases.

Laryngitis, croup, spasm of the glottis, and *œdema glottidis*, are considered very briefly. With respect to these affections, the author does not offer any opinions particularly novel. In treating of tracheitis and bronchitis, however, he advances views which are at variance with those commonly entertained. Inflammation limited to the trachea is generally thought to be extremely rare. It is usually regarded as almost always associated either with laryngitis or bronchitis. M. Beau, on the contrary, thinks that it is quite common, occurring much oftener than bronchitis; in fact, he considers the majority of the cases of so-called bronchitis, as purely cases of tracheitis. The reasoning on which this opinion is founded, is as follows: Inflammation of the bronchial mucous membrane, accompanied by an abnormal secretion of mucus, in more or less abundance, must, except the inflammation be quite limited, give rise to obstruction sufficient to occasion an emphysematous condition of the lungs, and, proportionately, dyspnœa. Under these circumstances, too, râles will

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[1] *Vide* Physical Exploration, etc., by the reviewer.

invariably be found over the chest. If, therefore, when inflammation is manifestly seated somewhere in the air-passages, there are no thoracic râles, no evidences of emphysema, and no dyspnœa—assuming the larynx to be unaffected—the affection is tracheitis.   The presence of tracheal râles is not necessary to the diagnosis ; for, owing to the size of the trachea, and the facility with which the mucus is expelled from this situation, in ordinary tracheitis, râles are not developed.   It is clear that on these conditions bronchitis does not exist in a large proportion of the instances in which its existence is assumed by medical practitioners. The reasoning by which the author sustains his position is purely theoretical. Will it bear critical analysis? We trow not.   That bronchial râles are often wanting in cases of so-called bronchitis, will be admitted.   Probably it oftener happens that they are wanting at the particular moments when auscultation is practised, than that they are altogether absent, for we know that they disappear and reappear at different times, being rarely constant during any period of the disease. But every practical auscultator must admit that bronchial râles are not infrequently more or less abundant in cases of so-called bronchitis, without the physical evidence of emphysema, and without dyspnœa. M. Beau assumes that these three diagnostic points are generally associated.   This assumption is certainly disproved by clinical facts. Bronchial râles, unusually constant and present in a marked degree, do not necessarily involve any appreciable embarrassment of respiration.   He accounts for the supposed necessity of dyspnœa in bronchitis, thus : the obstruction from the presence of mucus in the bronchial tubes, is overcome by the inspiratory movements ; while expiration, which is effected mainly by the force of elasticity, is insufficient for the free expulsion of air, which consequently accumulates and reacts upon the vesicles. This reasoning will again come up under the head of emphysema. We may simply state here that M. Beau overlooks the fact of the inspiratory being the powerful act only during tranquil respiration.   With muscular co-operation, either voluntarily or instinctively, to overcome an obstacle in the air-passages, the expiratory exceeds the inspiratory act, in force, by about one-third, according to the experiments carefully made with reference to this point, by Mr. Hutchinson.   We cannot stop to discuss this subject, more than others, at length. The impartial reader, we think, here, as in other portions of the work, cannot fail to be struck with the facility with which the author arrives at pathological conclusions, not less important than novel.

In treating of asthma, M. Beau ignores entirely the hypothesis of spasm advocated by M. Amédée Lefévre, in a prize essay, published in 1834, and generally adopted by medical writers.   He recognizes as the sole pathological condition productive of asthmatic paroxysms, the presence of tenacious mucus in the bronchial tubes.   To sustain this view, he is obliged to deny that an attack of asthma ever occurs instantaneously, and to assert that in all cases it is followed by more or less expectoration.   That an asthmatic paroxysm often ends before any expectoration takes place, he cannot but acknowledge, but in such cases he insists that the obstructing mucus is removed from the small into the larger tubes, where it ceases to interrupt the passage of air, and is subsequently expectorated.   According to this view, asthma is always neither more nor less than bronchitis affecting the smaller tubes.   We incline to the opinion that in the statement just made, the author is correct ; but that the dyspnœa in asthma is due exclusively to the presence of mucus, seems to us improbable in view of the small quantity expectorated even after some violent attacks.   The quantity of mucus, in other words, is insufficient to account

for so much embarrassment of respiration. The theory that the latter arises exclusively from spasm, on the other hand, is questionable. There remains a pathological condition which M. Beau does not recognize, viz., swelling of the bronchial mucous membrane. We may analogically infer that swelling in the smaller tubes sufficient to occasion dyspnœa more or less urgent, may occur suddenly, and last for a few hours only, just as we observe it to take place in the nostrils. This appears to us the most rational explanation of asthma ; and, as thus regarded, the affection, is, as M. Beau supposes, neither more nor less than bronchitis affecting the smaller tubes. We speak now of asthma occurring irrespective of emphysema.

The author passes from asthma to the affection just named, viz: emphysema. He treats only of the vesicular variety of emphysema. The theory of the mechanism of its production which he advocates is, that it is due to the reaction of the air within the vesicles upon the walls of the latter, when, from the presence of tenacious mucus, there is obstruction in the bronchial tubes. This reaction, of course, occurs in consequence of obstruction to the current of air in expiration; but the dilatation of the cells does not result from the difficulty of effecting the expulsion of air by the expiratory movements in respiration so much as from the existence of obstruction in connection with paroxysms of coughing. To quote the author's words—

"The accumulation of air depends on the fact that, being forcibly acted on by the movements incident to coughing, and with difficulty overcoming the obstacle afforded by the presence of mucus in the bronchial tubes, it reacts on the vesicles, which it dilates." (P. 146.)

Now, if the intensity of the respiration be increased, either voluntarily or instinctively, as we have seen, the expiratory is a more forcible act than the inspiratory. But, however this may be as regards respiration, there can be no question that the force with which the air is expelled with the act of coughing is vastly greater than that with which the air is inspired. How, then, can a collection of mucus furnish an obstacle which the violent expiratory effort in coughing with difficulty overcomes, but which does not obstruct the current of air in inspiration? This question suffices to show the absurdity of the theory. But, were we to pursue the subject further, it would be clear from the arrangement of the bronchial tubes, consisting of a series of branching cylinders successively diminishing in caliber, that a movable obstacle, like a plug of mucus, is likely to produce an obstruction to the passage of air much greater in inspiration than expiration. As pointed out by Dr. Gairdner, in the former case the mucus, moving in a direction from the larger to the smaller tubes, acts like a ball-valve, and limits or prevents the entrance of air into the cells over a greater or less number of lobules; while in the latter case, moving in an opposite direction, the tendency is to a removal of the obstruction. The ingenious theory of Dr. Gairdner, which attributes the origin of emphysema to collapse of more or less of the pulmonary lobules, produced by bronchial obstruction in the manner just mentioned, is not alluded to by M. Beau. Both theories agree in considering bronchial obstruction as the point of departure for the production of this lesion; but it is curious that precisely opposite effects of the obstruction are described by these two authors. M. Beau says the result is a dilatation of the cells from the difficulty of the egress of air during the expiratory efforts incident to coughing. Dr. Gairdner says that the obstruction affecting chiefly the ingress of air in inspiration, collapse of more or less of the lobules ensues, and consequently an abnormal expansion of the remaining lobules from the atmospheric pressure. So far as the comparative merits of

the two theories are concerned, we do not see how any one can hesitate to give preference to that of Dr. Gairdner.[1]

M. Beau devotes considerable space to the discussion of the conclusion to which Louis was led by his researches, viz., that the production of emphysema does not necessarily or generally involve the pre-existence of bronchitis, but that it is a primary or spontaneous lesion occurring irrespective of any mechanical agency, being due to an abnormal condition of the force which presides over the development of hollow organs. In contending for the dependence of emphysema on bronchitis in the majority of instances, we must think M. Beau is correct, albeit this opinion is opposed by so accurate and truth-loving an observer as Louis. In not accepting the conclusion at which the latter arrives relative to this point, we are not guilty of the presumption of doubting either his capacity or honesty. In a large proportion of the cases of emphysema met with in the wards of a hospital, the affection has existed for a greater or less period, often for many years, having been perhaps developed in childhood. Under these circumstances, it is often impossible to obtain reliable information as to whether the symptoms of the emphysema or those of bronchitis had precedence. That the latter precedes in certain instances we know, for it has occurred to us, within the past few years, to have witnessed the development of emphysema, as it were under our eyes, in two patients whom we had repeatedly examined while suffering from persisting bronchitis, before dilatation of the vesicles had ensued.

Finally, on the subject of emphysema, the author makes a remark which is worthy of being borne in mind, with reference to the portion of the lung most prone to become affected with this lesion. Pathologists generally are agreed that the superior and anterior portion is especially apt to be found in an emphysematous condition. M. Beau thinks this is not certain, inasmuch as the dilatation of the cells in the posterior portion cannot be ascertained by the gross appearances, in consequence of the existence here of hypostatic congestion.

Under the head of pneumonia we meet with nothing which, from its novelty, claims notice. The same is true of pleurisy, pneumo-hydrothorax, gangrene of lung, pulmonary apoplexy, and œdema, except that in the latter affection the author differs from pathologists in thinking that the serous effusion takes place, not into the air-cells, but into the connecting areolar tissue.

Under the head of pulmonary tuberculosis, the only point on which we desire to comment is the significance of a prolonged expiration. It is well known that our lamented countryman, James Jackson, Jr., first directed attention to the importance of studying the expiratory murmur. He showed that a prolongation of the expiratory sound was frequently a sign of the presence of tubercles, and that, in the development of the bronchial respiration, the abnormal change is manifest in the relative direction of this sound. Jackson studied only the characters of the expiration which pertain to duration and intensity; those relating to quality, and especially pitch, were left for other observers. M. Beau states that a prolonged expiration much oftener denotes a cavity than the presence of crude tubercles, and he adds:—

"I am not aware of any sign which may enable us to distinguish the prolonged expiratory murmur due to crude tuberculization from that due to cavities."

[1] For Dr. Gairdner's views, vide *On the Pathological Anatomy of Bronchitis and the Disease of the Lung connected with Bronchial Obstruction.* Edinburgh, 1850. For review of this work, vide *British and Foreign Medico-Chirurgical Review,* April, 1853. These views are not unknown in France. They are adopted by Valleix in the last edition of his *Guide du Praticien.*

This distinction may be made without difficulty, if the description which we have elsewhere given of the expiratory sound in the bronchial and cavernous modifications of respiration are based on correct observation.[1] According to this description, in bronchial respiration the expiratory generally exceeds the inspiratory sound in intensity, and more especially in pitch. The reverse obtains in the cavernous respiration; the expiratory sound is less intense and lower than the inspiratory. We have here a means of discrimination which, if reliable, is certainly of practical importance. Theoretically, M. Beau could not very consistently admit this distinction, inasmuch as he refers the origin of the cavernous as well as of the bronchial and vesicular respiratory sounds to the glottis.

The number and character of the peculiar views entertained by M. Beau, pertaining to auscultation as applied to the respiratory organs, prepares the reader to expect a similar series in the portion of the work which treats of the heart. In this respect, the reader's expectation is not disappointed. The author contends for theories of the mechanism of the movements and sounds of the heart in health and disease, strikingly at variance with the conclusions which others have deduced from the results of investigations bestowed on this subject. We shall devote the remainder of this article to a brief notice of these theories.

The question whether the apex of the heart elongates or contracts during the systole of the ventricles, has long been mooted. According to Vesalius, and other of the older anatomists, the vertical diameter of the organ is increased. This was the opinion of Harvey. Subsequent observers have generally described the systolic contraction as extending to all the diameters.

Some, however, have been convinced that this description is incorrect, and that the heart is lengthened during the systole of the ventricles.

Doctors Pennock and Moore were led to this conclusion by their experiments made in Philadelphia in 1839. If we are correctly informed, Professor Dalton, of New York, has arrived at the same conclusion, and is accustomed to verify its correctness in his vivisections performed in the class-room. M. Beau believes that the heart is shortened during the ventricular systole. It would seem that this is a question which might be easily settled by observation. It is manifestly to be thus settled, and not by *à priori* reasoning from the direction which the muscular fibres are found to pursue on dissection of the heart. May it not be the case that different species of animals, or different individuals of the same species; or, again, the heart of the same individuals under different circumstances pertaining to the circulation, differ in this respect, shortening occurring in some and elongation in other instances?[2] This suggests itself as affording an explanation of a singular discrepancy of opinion on a matter respecting which, if there be uniformity in fact, one would think all observers should be agreed.

Most observers agree in attributing the impulse of the heart in the præcordia to the movements of the apex during the ventricular systole. M. Beau, however, holds a different opinion. He considers the impulse as occurring during the ventricular diastole. This theory, which may be distinguished as

[1] *Vide* Prize Essay on Variations in Pitch of Percussion and Respiratory Sounds, 1851; and Physical Exploration, etc., 1856.

[2] We can easily imagine that this is a difficult point of observation, if small animals are employed for the experiments, such as the frog or birds; and in larger animals the spiral movement of the heart probably renders it somewhat difficult to determine whether shortening or elongation occurs.

the theory of diastolic impulse, enunciated by M. Beau more than twenty years ago, has a few supporters in Europe, and in this country has found an able advocate in Professor Alfred Stillé. The theory involves other peculiar views concerning the mechanism of the heart's action. According to M. Beau, the ventricular diastole is due to the systolic contraction of the auricles. The ventricles, after their systole, are passive; they do not dilate, as is generally supposed, either from their elasticity or as a result of the contraction of certain of the muscular fibres which enter into the composition of their walls, but they are dilated solely by the distending force derived from the current of blood impelled into their cavities, by the contraction of the auricles. The auricles, thus, are in fact, the active agents in the dilatation of the ventricles. The force of the current of blood driven into the ventricle by the auricular contraction, elongates the latter, and gives to the organ the movements against the thoracic wall which occasion the impulse or beating in the præcordia.

The diastolic theory involves an order of succession in the movements of the auricles and ventricles different from that generally supposed to take place. M. Beau takes as a point of departure the contraction of the auricles. The auricular systole causes the diastole of the ventricles, and the ventricular contraction immediately follows. Then occurs the period of repose of the organ; viz., the period which elapses from the completion of the ventricular, to the commencement of the next auricular systole. According to the commonly received descriptions, the diastole of the ventricles immediately succeeds their contraction, and the period of repose is supposed to be after their diastole, before the succeeding systole. The distinctive feature in the theory of M. Beau, so far as concerns the order of succession of the movements, relates, then, mainly to the occurrence of the diastole of the ventricle. According to this theory, it occurs just *before* the ventricular systole, and is *preceded* by a period of repose. According to the current doctrine, it occurs immediately *after* the ventricular systole, and is *followed* by a period of repose.

We have called these views of M. Beau *a theory.* The points, however, which have just been mentioned are not properly subjects for theoretical discussion in the vulgar acceptation of this term. They are points to be settled by simple observation. Does the heart, as determined by the sight and touch, perform movements adequate to the production of the well-known impulse in the præcordia during the ventricular systole or diastole? Do the senses afford evidence that the ventricles dilate immediately after they contract, prior to any apparent contraction of the auricles, or does this dilatation take place after the lapse of a certain time, and do the auricles previously contract? These questions are to be answered by an appeal to facts ascertained by exposing the heart to view in inferior animals during life, and by witnessing its movements in the few remarkable instances of malformation in which either this organ has been situated exterior to the thoracic wall, or, from deficiency of the sternum and costal cartilages, it has been open to inspection *in sitû.* Several instances of these two kinds of malformation are recorded in the annals of medicine; and we need not remind those of our readers who have bestowed any attention on this subject, that numerous experiments have been made on a grand scale in London, Dublin, Edinburgh, and in this country.[1] To repro-

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[1] The reader will find these experiments detailed in full in *Hope's Treatise on Diseases of the Heart*, American Edition, with Additions, by Dr. Pennock. Also a *résumé* in the last edition (1854) of the *Traité Pratique D'Auscultation, par Barth et Roger.* The latter work contains a full discussion of the theories of M. Beau.

duce these experiments and observations, or even to present a digest of the results, would occupy too much space, and is foreign to our present purpose. With some discrepancies relating to points of more or less importance, they furnish the basis of the doctrine relating to the impulse of the heart and the order of succession in the movements of its parts, which are commonly received, and are opposed to the views of M. Beau. How does the latter sustain his theory? He adduces the results of experiments performed by himself on frogs, rabbits, dogs, and birds. As regards the animals selected, and the manner of performing the experiments, there is room for criticism; but, waving this, how are we to account for the discrepancy of his results with those of other observers? Evidently by the same rules of evidence which should influence a judge or jury in courts of law when conflicting testimony is rendered by different witnesses. Assuming that a witness is perfectly honest, if it appear that he is interested or prejudiced, his testimony carries but little weight. Again, if there are numerous witnesses, and the great majority concur as to certain points, this is a fair ground for presuming that the few who offer rebutting testimony are in the wrong. Judged by these rules, the results of M. Beau's experiments are to be received with distrust. They were made, in great part, to confirm theoretical views which he had advanced many years before. The experiments made by the London, Dublin, and Philadelphia committees were made, on the other hand, in the presence of those who were not thus committed. Numerically, the testimony in behalf of the results obtained by the latter greatly preponderates.

In saying that the foregoing points pertaining to the movements of the heart are to be settled by simple observation, we would not be understood to attach no importance to inferential conclusions. In fact, the incorrectness of the theory of diastolic impulse, and the theoretical views which it involves, may, we conceive, he conclusively shown without resorting to an ocular examination of the heart in action. The impulse of the heart, and that of the arteries situated near the central organ of the circulation, are found to be synchronous. Now, admitting, as M. Beau states (and in this respect the observations of others concur), that the systole of the ventricle succeeds very quickly that of the auricle, if the heart's impulse be due to the latter, there should be a distinctly appreciable interval between it and the diastolic impulse of the arteries situated ever so near the heart. Inferentially, it is quite inconceivable that the amount of muscular structure contained in the valves of the auricles, is sufficient to propel the blood into the ventricular cavities with a degree of force adequate to produce the præcordial impulse, more especially when the latter becomes as violent as it frequently does in palpitations. If the auricles were indeed capable of contracting with the force requisite to account for the heart's impulse, it is inconceivable that regurgitation into the veins should not habitually occur sufficiently to give rise to venous pulse in the jugular veins at least, such as we find does occur when regurgitation through the left auriculo-ventricular orifice takes place during the ventricular systole, owing to insufficiency of the tricuspid valves. Again, it is irrational to suppose that, after the systole, the ventricle remains firmly contracted until a forced dilatation results from the action of the auricle. The ventricles and auricles communicate somewhat like chambers with folding doors, which admit of being opened with the slightest force, but only in one direction. What is to hinder the passage of blood from the auricle into the ventricle by the force of gravitation, added to that derived from the column of blood pouring into the former from the veins, except the ventricle be closed by an active contraction of its muscular walls? This persisting contraction pre-

cludes any period of muscular repose. M. Beau meets this objection by assuming that the ventricular systole is succeeded by a state of tonic contraction adequate to resist the ingress of blood until it is poured into the cavity by the auricular contraction. This, besides being hypothetical, is highly improbable.

The study of the mechanism of the normal sounds of the heart has occasioned great diversity of opinion, and the subject must be considered still open for discussion and fresh researches. This remark applies more especially to the first sound. Most physiologists, we believe, are agreed as regards the dependence of the second sound on the arterial valves; but admitting the latter to be concerned in their production, the mode in which they are produced, that is, the physical principles involved, is not fully settled. Among the twenty-seven theories of the heart's sounds, enumerated by MM. Barth and Roger, the theory of M. Beau is included, and perhaps is as unsatisfactory as any in the list. But it is to be considered that in theorizing on this subject, M. Beau labours under the disadvantage of being obliged to conform to his theory of the movements of the heart. The first sound, according to the theory of diastolic impulse, is not synchronous with the systole, but with the diastole of the ventricles. M. Beau attributes it to the shock caused by the current of blood driven by the auricular contraction into the cavity of the ventricle, and striking abruptly against the inferior portion of this cavity. Without discussing the intrinsic merits of this explanation, it is sufficient to say that it cannot be true if the theory of diastolic impulse be false. Its claims are thus summarily disposed of. The second sound M. Beau attributes to the column of blood arising at the auricles from the veins, and impinging against the auricular walls. This hypothesis strongly illustrates the difficulty with which an explanation of the second sound is made to harmonize with the author's theory of the heart's movement. It would be, however, needless to subject the arguments adduced in its behalf to critical analysis, or to show its improbability and inadequateness, for it is disproved by the facts determined by repeated experiments, establishing beyond a reasonable doubt, that the second sound is produced at the arterial orifices of the ventricles, and that the semilunar valves are concerned in its production.[1]

M. Beau recognizes certain accessory normal sounds, which are generally combined with the first sound of the heart, and serve to reinforce and to modify its character to some extent. One of these accessory sounds is the ringing intonation, called by Laennec the *cliquetis métallique*. M. Beau attributes this to the striking of the apex of the heart against the thoracic wall. We believe this to be the correct explanation. If the ribs are pressed with the finger, while the stethoscope is applied at a situation not far removed from the point of percussion, the note elicited has an amphoric quality. We can understand that this intonation will be marked especially when the stomach is distended with gas. Another accessory sound is the *bruit musculaire*. M. Beau attributes this to the ventricular systole which succeeds the auricular contraction so quickly as to cause this *bruit* to be incorporated with the first sound.

M. Beau arranges the rhythmical succession of the heart sounds in a manner peculiar to himself. He regards the first and second sounds as equal in duration, and the length of the interval between the second and the first sound as the same as either of the sounds. Musically expressed, the succession of

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[1] *Vide* Experimental Researches, contained in *Hope's Treatise*, Amer. ed.

sounds is represented by triple time, the sounds being indicated by two crotchets, and the silence by a rest.

Connecting with each of the beats in the bar (according to this representation), the movements of the heart which belong to it, after the theory of M. Beau, the result is as follows: The first beat or crotchet embraces the contraction of the auricles, the dilatation of the ventricles, and the ventricular systole. These movements, in other words, are all involved in the first sound. The second beat, or sound, embraces simply the dilatation of the auricles. During the *rest*, or silence, repletion of the auricle takes place—the ventricles remaining contracted and empty.

In the foregoing arrangement, the author differs from nearly all other observers as respects the relative duration of the first and second sounds, the former being generally reckoned nearly twice as long as the latter. He overlooks the brief period of silence between the first and second sound, and he estimates the period of silence following the second sound at a higher fraction than is usually employed, viz., at one-third.

The abnormal sounds are divided into the extra and intra-cardiac murmurs. The extra-cardiac or attrition sounds are considered very briefly, the author, under this head, presenting simply a succinct statement of well-known facts. As respects the intra-cardiac murmurs, however, he indulges his fondness for original views, and, in fact, his theories of the movements and normal sounds of the heart render it necessary to adopt explanations of these murmurs at variance with the opinions commonly entertained. The reader can hardly fail to recognize in the portion of the work treating of the abnormal, or in that in which the normal sounds are considered, an effort of adaptation to the theories pertaining to the movements of the heart. This adaptation demands not only deviation from the opinions generally received, but a denial of certain pathological facts usually considered as established.

M. Beau attributes the production of intra-cardiac, or, as they are commonly called, bellows murmurs, to an exaggerated friction arising from a want of proportion between the volume of the current of blood and the size of the cardiac passages. This want of proportion involves positive or relative contraction of one or more of the orifices.

A bellows murmur, as every practical auscultator is aware, is associated in the vast proportion of instances with the first sound of the heart. This sound, according to the doctrine of the diastolic impulse, is caused by the auricular contraction. To be consistent, therefore, with this doctrine, it is necessary to assume that the murmur is caused by the passage of blood in its natural course from the auricle to the ventricle. Of the several orifices, the left auriculo-ventricular is oftenest the seat of lesions. Now, assuming that the blood passes from the auricle, after the apex impulse during the ventricular diastole, according to the doctrine generally believed, a murmur generated by the current in this direction, it is evident, will in point of time be associated with the second and not the first sound. Hence it follows that, according to this doctrine, the murmur incident to disease of the mitral orifice, and accompanying the first sound, must be due to regurgitation. But it does not suit M. Beau's theories of the movements and normal sounds to attribute the murmur, under these circumstances, to regurgitation. This explanation is,

in fact, incompatible with the doctrine of diastolic impulse. How does M. Beau overcome this difficulty? By asserting that regurgitation takes place only exceptionally, *i. e.*, in a very small proportion of cases. He says :—

" I certainly do not deny, absolutely, that a reflux of blood into the auricle may take place, and a murmur be caused by this reflux; but I think that this occurs only in some exceptional cases. In fact, it is hardly conceivable that the blood should be forced by the ventricular contraction into the auricle, when it finds an easy egress by the arterial orifice ; for this reason I think that the reflux into the auricle occurs only in cases in which contraction of the arterial orifice complicates insufficiency of the auriculo-ventricular valves, obstructing the passage of the blood from the ventricle into the artery." (P. 300.)

These assumed pathological facts are so extraordinary that the author was bound to adduce data for their support, aside from the consideration that they are necessary to sustain his physiological theories. We have a right to demand evidence gathered in the dead-room for statements directly opposed to the opinions of other observers. In the absence of this evidence, the author cannot complain that the reader accounts for the statements on the principle of adaptation to which we have alluded; in other words, the author believes the statements to be facts, because otherwise his theories of the normal sounds and movements of the heart will be invalidated.

A murmur accompanying, not following, the first sound, M. Beau is obliged to refer always to the auriculo-ventricular orifice. Conformity to his physiological theories requires this. If produced by the ventricular systole, at the arterial orifices, they must occur after the first sound and impulse. This he asserts to be the case. But this assertion is certainly disproved by clinical experience.

Again, a murmur associated with the second sound, according to clinical observation, is sometimes, although rarely, due to the current of blood from the auricle to the ventricle. This, of course, M. Beau cannot admit, inasmuch as the theory of diastolic impulse involves the passage of blood in that direction synchronously with the first sound. He denies, therefore, that a bellows murmur with the second sound ever originates at the auriculo-ventricular orifice, and asserts that it always depends on regurgitation from the aorta.

It is well known that, in determining the seat of intra-cardiac murmurs, considerable importance is attached by auscultators to the situation where they are heard with a maximum of intensity, and the direction in which they are furthest propagated. If any rules in physical exploration are well established, it is that these two points are entitled to confidence as a guide in the localization of valvular lesions. We can testify that, in a clinical experience considerably extended, we have found them reliable. But M. Beau cannot, of course, admit that an arterial murmur with the first sound is heard loudest over the arterial valves, and transmitted upward by the vessels, while it is perhaps quickly lost as the stethoscope is carried over the body of the heart toward the apex. He cannot admit this fact, demonstrable as it is at the bedside, because it is incompatible with the theory of diastolic impulse. According to this theory, the blood is not propelled into the arteries by the movement which causes the first sound. Hence, as just stated, he denies that a murmur produced at the arterial orifices ever accompanies the first sound.

The length to which this review is already extended precludes notice of the section devoted to the individual affections of the heart. The points, however, embraced in this section, inviting criticism, mainly relate to the application of the theoretical views to which we have referred in the foregoing remarks. For

the same reason, we must forego any critical notice of the concluding portion of the work, which treats of auscultation as applied to the bloodvessels. An analysis of the latter would develop peculiar doctrines not less than the portions which we have passed in review, but they have neither the same interest nor importance.

As before stated, the object of this work is to reproduce and bring together opinions of the author heretofore submitted in numerous papers distributed in different medical periodicals. And it is a volume truly remarkable for a collection of opinions which, for the most part, although long before the profession, and advocated by the author with great earnestness and ability, have been adopted by others only to a very limited extent.

Judged by the character of the work, the author's mental constitution is marked by certain peculiarities, which are often enough observed, but rarely in connection with so much industry and real ability. A pruriency for originating novel views appears to be the leading characteristic. Having formed a new theory, it is irrevocable, so far as his own belief is concerned. Facts which are incongruous, however stubborn, are less obstinate than his faith, and only serve to task his ingenuity in endeavouring to effect a compromise; hence he finds it easier to modify or reject the former, than to alter or abandon the latter. A clinical observer, he sees exclusively through the medium of the preconceived notions which he has originated. The work is both interesting and valuable in a psychological point of view, exemplifying that talent, zeal, and honesty are not sufficient for the investigation of truth, and that, without the well-balanced mind which belongs to the true philosopher, these mental qualities may serve to retard instead of promoting the advancement of science. A. F.